POSTOPERATIVE VOCAL CORD PARALYSIS IN PAEDIATRIC PATIENTS

Reports of Cases and a Review of possible Aetiological Factors

BY

M. R. SALEM, A. Y. WONG, V. C. BARANGAN, R. F. CANALIS, M. H. SHAKER AND A. M. LOTTER

SUMMARY

Two cases are reported of recurrent laryngeal nerve paralysis that followed thoracic manipulations in paediatric patients. Possible aetiological factors in relation to anaesthesia and operation in paediatric and adult patients are reviewed. These include: forceful endotracheal intubation; direct surgical trauma; compression of the nerve in the neck by a tracheal cuff, or a large endotracheal tube, in the thorax by changes in the size of the great vessels; stretching of the nerve as a result of traction on distant organs; toxic effects of substances present in endotracheal tubes or formed as a result of sterilization; and the presence of a concomitant upper respiratory tract infection.

Vocal cord paralysis following certain neck operations is a well-recognized complication. However, it is less appreciated following thoracic or abdominal operations. The present report illustrates the occurrence of this postoperative complication in two paediatric patients in relation to thoracic manipulations, and includes a review of possible aetiological factors.

CASE 1

A newborn infant with the diagnosis of tracheo-oesophageal fistula underwent gastrostomy, right thoracotomy and closure of the fistula under uneventful endotracheal halothane anaesthesia. Anastomosis of the proximal and distal ends of the oesophagus could not be performed at this stage because of the wide gap between the two segments. Over the ensuing 8 weeks, the patient underwent oesophageal dilatations to enlarge and lengthen the proximal oesophagus.

At 8 weeks of age, the patient underwent right thoracotomy and oesophageal anastomosis under nitrous oxide, oxygen halothane anaesthesia. Endotracheal intubation was performed smoothly with a No. 18 French Cole endotracheal tube and facilitated with an injection of suxamethonium. The patient woke up promptly after the operation was completed, and was extubated. Stridor and hoarseness were noted in the second hour postoperatively. Gradually, supraclavicular and intercostal retractions became apparent, especially during crying. Laryngeal oedema was suspected and dexamethasone 2 mg was given intravenously and the patient was placed in a tent with 40 per cent oxygen and high humidity, but the stridor and hoarseness persisted. Laryngoscopy was performed and revealed complete absence of movement of the right vocal cord, which was in a midline position, while there was satisfactory movement of the left cord. There was no evidence of laryngeal oedema or trauma. Over the next 48 hours, the patient gradually improved except for slight hoarseness. Thereafter, he had an uneventful recovery. The cords were visualized 10 days following the operation and normal movement of both cords was seen.

CASE 2

A 2-year-old child was admitted to the hospital with the diagnosis of oesophageal stricture, secondary to lye ingestion. Oesophageal dilatation was attempted under general anaesthesia and a feeding gastrostomy was performed. Subcutaneous emphysema was noted in the neck postoperatively and oesophageal rupture was suspected. Purulent material was drained from the superior mediastinum and chest drainage tubes were inserted on the right side on the third postoperative day.

Three months later, and after the perforation had healed, colon bypass of the oesophagus and pyloroplasty were performed in a 6-hour operation via midline abdominal and small transverse left supraclavicular incisions. Anaesthesia was induced with halothane, nitrous oxide and oxygen. Intubation was easily accomplished with a size 24 clear plastic (polyvinyl chloride) endotracheal tube, following suxamethonium incisions. Anaesthesia was induced with halothane, nitrous oxide and oxygen. Intubation was easily accomplished with a size 24 clear plastic (polyvinyl chloride) endotracheal tube, following suxamethonium administration. Tubocurarine was injected intermittently as required. At the termination of the procedure, the effect of tubocurarine was reversed with an atropine-neostigmine mixture and the patient was finally extubated.

* University of Chicago, Pritzker School of Medicine, Department of Otolaryngology, 950 East 59th Street, Chicago, Illinois 60611.
† Northwestern University School of Medicine, Department of Otolaryngology, 303 East Chicago Avenue, Chicago, Illinois 60611.
Inspiratory stridor was noted shortly after extubation which gradually worsened. Direct laryngoscopy showed no evidence of laryngeal oedema or trauma, but the cords were fixed in a midline position. Vigorous ventilation resulted in a downward displacement of the cords and complete respiratory obstruction. Five hours later, tracheostomy was performed after the trachea had been reintubated. In the first week, postoperatively, the patient continued to have difficulties with swallowing fluids with “spill over” into the trachea.

Repeated laryngoscopic examinations showed no change in the bilateral vocal cord paralysis. Left arytenoidectomy was done two months later and resulted in a slight improvement, but the function of both cords never returned.

DISCUSSION

Postoperative vocal cord paralysis may be due to mechanical or neurogenic factors. Mechanical laryngeal injury such as dislocation and subluxation of the cricothyroid or cricoarytenoid joints may result from traumatic endotracheal intubation. Such an injury does not seem to have played a role in the occurrence of vocal cord paralysis in the cases presented, since there was no evidence of laryngeal trauma on laryngoscopic examination.

The occurrence of vocal cord paralysis in both cases is most probably related to surgical manipulations in the thorax, resulting in recurrent laryngeal nerve injury. In the first case, the lesion occurred in the same side of the chest that was explored. In the second case, bilateral recurrent laryngeal nerve paralysis was apparently due to the manipulations exerted in the thorax and lower part of the neck during bypassing the oesophagus by the colon. The occurrence of paralysis in the first case cannot be due to severance of the right recurrent nerve, since there was a rapid return of function 10 days later. This could be explained on the basis of traction applied to the nerve during the operation. Unilateral vocal cord paralysis in newborn infants is known to be followed by rapid recovery in a high percentage of cases (Ballenger, 1969).

A recent Russian article has indicated a surprisingly high incidence of recurrent nerve paralysis accompanying thoracic operations in adults and children (Podgaetskly, 1967). These figures were reported: 8.1 per cent after pneumonectomy, 1.7 per cent after lobectomy, and 1.1 per cent following segmental resection of the lung. Following repair of coarctation of the aorta, 7.5 per cent of the patients developed recurrent laryngeal nerve paralysis and the incidence was 6 per cent after repair of patent ductus arteriosus.

Direct trauma during the surgical procedure to one or both nerves is still the commonest cause of postoperative recurrent laryngeal nerve paralysis and, hence, the relatively high incidence following neck and thoracic operations. However, vocal cord palsy has been reported in relation to abdominal (Yamashita et al., 1965) and other surgical sites far from the anatomical course of the recurrent laryngeal nerves (Yamashita et al., 1965; Hahn, Martin and Lillic, 1970; Holley and Gildea, 1971). Subsequently several theories have been suggested to explain the occurrence of this phenomenon in relation to anaesthesia.

Hahn, Martin and Lillic (1970) related the occurrence of unilateral paralysis in their five adult patients to the irregular inflation of the endotracheal cuff. They suggested that unequal pressure could be exerted within the larynx by an endotracheal cuff placed just below the cords, and could compress the nerve endings at that side against the thyroid cartilage, resulting in unilateral paralysis. Although overexpansion of the endotracheal cuff is feasible, it does not seem to play an important role. In both our cases, non-cuffed endotracheal tubes were used. Furthermore, in the recent report of Holley and Gildea (1971) on four adult patients who developed bilateral vocal cord paralysis, they indicated that the endotracheal cuff was inflated only enough to provide an airtight seal around the endotracheal tube.

In an autopsy performed on one of the cases reported by Holley and Gildea (1971), myelin degeneration was seen in both vagi. In view of their finding, damage from ethylene oxide sterilization of Portex endotracheal tubes was incriminated as a possible cause of vocal cord paralysis. Toxic substances capable of causing tissue injury have been recently discovered in endotracheal tubes (Guess and Stetson, 1968). It was also found that plastic endotracheal tubes sterilized with ethylene oxide may exert toxic effects because of the formation of ethylene chlorhydrin in tubes previously sterilized by gamma radiation (Cunliffe and Wesley, 1967). If the endotracheal tubes are not adequately aerated, chemical burns and ulceration to the tissues may result. Because of this, the use of sterile, disposable endotracheal tubes has been recommended (Rendell-Baker, 1968).

Although the theory that toxic effects following gas sterilization may cause vocal cord paralysis,
as suggested by Holley and Gildea (1971), is attractive, it does not provide a satisfactory explanation for the cord paralysis that occurred in all their reported cases. In one of their patients thyroidectomy was performed and it is difficult to exclude the possibility of trauma to the nerves under such circumstances. In another patient, there was a pre-existing history of sustained unilateral vocal cord paralysis following a previous thyroidectomy and the patient had a history of hypertensive cardiovascular disease, a condition that may mechanically cause palsy of the left recurrent laryngeal nerve. In a third patient, laryngeal stridor was present pre-operatively, the cause of which was not sought.

Paralysis of the recurrent laryngeal nerves may occur in a wide variety of conditions. It is usually due to disease in adjacent tissues, to thyroidectomy or, less often, to disorders of the nervous system. In a proportion of cases no cause can be found, a condition often termed “idiopathic recurrent laryngeal nerve paralysis” (Williams, 1959). In view of recent reports incriminating endotracheal intubation, it has been proposed that perhaps some patients with “idiopathic paralysis” might have had general anaesthesia with a cuffed endotracheal tube (Hahn, Martin and Lillie, 1970). If endotracheal intubation is a definite contributing factor, one may expect that there has been a significantly higher incidence of “idiopathic paralysis” in recent years. However, this is not the case. In a series of 181 patients with recurrent laryngeal paralysis, Williams found that the incidence of idiopathic paralysis was 36 per cent of all cases whilst in 1932 New and Childrey assessed the incidence of idiopathic paralysis as 33 per cent of all cases of recurrent laryngeal nerve paralysis.

One postulated mechanism for the occurrence of palsy in certain pathological conditions, such as left ventricular failure and mitral stenosis, is worth mentioning. It was shown that in these situations the left recurrent laryngeal nerve is actually compressed between the left pulmonary artery, aorta, and the obliterated ductus arteriosus (King, Hitzig and Fishberg, 1934). They reported the history of one patient, who had left recurrent laryngeal palsy due to left ventricular failure; it was found at autopsy that the portion of the nerve which lies between the arch of the aorta, left pulmonary artery, and the obliterated ductus was definitely constricted and discoloured. Marked degenerative changes in the same portion of the nerve were noted on microscopic examination. Dilatation of the pulmonary artery or aorta may then result in compression of the left nerve which is prevented from being displaced to the right by the ligamentous arteriosus (King, Hitzig and Fishberg, 1934). Left recurrent paralysis may also occur in children with congenital heart disease (Ballenger, 1969). The mechanism of production of paralysis is possibly similar to that in left ventricular failure and mitral stenosis in adult patients. Alterations in the pressures and sizes of the great vessels are known to occur in anaesthetized surgical patients (Salem, Yacoub and Holaday, 1968). Whether a contributing factor or not, it is not known at present.

It is possible that traction on certain organs, such as the lower end of the oesophagus, although remote from the anatomical course of the nerve, may lead to its stretching with subsequent paralysis. This may be the explanation of paralysis occurring in relation to abdominal operations. In one report from Japan (Yamashita et al., 1965), all of the nine cases were associated with abdominal surgery. The long anatomical course of the nerve probably contributes to the relatively higher incidence of palsy of this, compared with other nerves. In this respect, it may simulate the abducens nerve, a nerve that is more frequently affected than any other intracranial nerve following lumbar puncture (Hayman and Wood, 1942). The left recurrent laryngeal nerve, being of greater length than the right, is more vulnerable to adjacent diseases, trauma and stretching.

Recurrent laryngeal paralysis has been reported in patients with upper respiratory infections (Clerf, 1953). It may also be caused by exposure to cold in a manner similar to that suggested for Bell’s palsy (Williams, 1959). In fact, both conditions have been reported to occur simultaneously (Faaborg-Anderson, 1954).

Whatever the cause may be (table I), anaesthetists must be aware of the possibility of vocal cord paralysis occurring in operations remote from the neck. Moreover, signs and symptoms of recurrent laryngeal nerve paralysis may simulate laryngeal oedema, a well-known complication related to endotracheal intubation in
POSTOPERATIVE VOCAL CORD PARALYSIS IN PAEDIATRIC PATIENTS

TABLE I

<table>
<thead>
<tr>
<th>Theories of the causation of postoperative vocal cord paralysis.</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. Mechanical laryngeal injury to the cartilaginous joints due to traumatic endotracheal intubation.</td>
</tr>
<tr>
<td>II. Recurrent laryngeal nerve paralysis.</td>
</tr>
<tr>
<td>Direct surgical trauma to the nerve in the neck or thorax.</td>
</tr>
<tr>
<td>Compression of the nerve.</td>
</tr>
<tr>
<td>In the neck: By endotracheal cuff or a large endotracheal tube.</td>
</tr>
<tr>
<td>In the thorax: By changes in the size of the great vessels.</td>
</tr>
<tr>
<td>Stretching of the nerve, as a result of traction on certain distant organs (e.g. lower end of the oesophagus).</td>
</tr>
<tr>
<td>Toxic effect of substances present in endotracheal tubes or formed as a result of sterilization.</td>
</tr>
<tr>
<td>Concomitant upper respiratory tract infection.</td>
</tr>
</tbody>
</table>

Infants. Laryngeal oedema usually responds to treatment with humidification, steroid therapy and spraying the cords with a dilute vasoconstrictor solution, while the response is minimal with vocal cord paralysis. Exaggerated respiratory activity, whether spontaneous or induced, may aggravate the already existing respiratory obstruction in both situations and must be avoided. Furthermore, infants with vocal cord paralysis may aspirate during swallowing because of their inefficient cough mechanism. In both our cases, the diagnosis was initially mistaken for laryngeal oedema, but excluded on laryngoscopic examination. In cases of persistent stridor, it seems more appropriate to inspect the larynx to verify the diagnosis, so that proper treatment can be given.

REFERENCES


PARALYSE POSTOPERATOIRE DES CORDES VOCALES CHEZ LE PATIENT PEDIATRIQUE: DESCRIPTION DE CAS ET REVUE DES FACTEURS ETIOLIQUES POSSIBLES

SOMMAIRE

Des auteurs décrivent deux cas de paralysie récurrente du nerf laryngé après manipulation thoracique chez des enfants. Ils présentent une revue des facteurs étiologiques possibles par rapport à l'anesthésie et l'opération d'enfants et adultes, dont: l'intubation endotracheenne forcée; le traumatisme chirurgical direct; compression du nerf dans le cou par la manchette trachéenne ou un grand tube endotracehénien, dans le thorax par des modifications de la taille des grands vaisseaux; l'extension du nerf par suite de la traction à distance; les effets toxiques de substances présentes dans le tube endotracehenien ou formées par suite de la stérilisation; et la présence d'une infection simultanée des voies respiratoires supérieures.

POSTOPERATIVE STIMMBANDLAHMUNG IN DER KINDERCHIRURGIE: CASUISTISCHER BERICH T UND ÜBERSICHT ÜBER MÖGLICHE ATIOLOGISCHE FAKTOREN

ZUSAMMENFASSUNG

Es werden zwei Falle von Recurrens-Lähmung im Anschluss an thorax-chirurgische Eingriffe bei Kindern vorgestellt. Mögliche atiologische Faktoren bezüglich Anaesthesie und Operation bei Kindern und erwachsenen Patienten werden erwogen. Dargestellt sind: Kraftrige endotracheale Intubation, direktes chirurgisches Trauma, Kompression des Nerven im Hals durch Trachealmanschette oder großen endotrachealen Tubus, im Thorax durch Größenveränderungen der Gefäße; Dehnung des Nerven durch Zug an entfernten Organen; toxische Wirkungen von Substanzen im Tubus oder entstanden durch die Sterilisation; ferner eine gleichzeitig bestehende Infektion der oberen Atemwege. 
PARALISIS POSOPERATORIA DE LA CUERDA VOCAL EN PACIENTES PEDIATRICOS.
COMUNICACION DE CASOS Y REVISION DE LOS FACTORES ETIOLOGICOS POSIBLES

RESUMEN
Son comunicados dos casos de parálisis del nervio laringeo recurrente después de manipulaciones torácicas en pacientes pediátricos. Son revisados los factores etiológicos posibles en relación con la anestesia y operación en pacientes pediátricos y adultos; estos incluyen la intubación endotraqueal forzada; trauma quirúrgico directo; compresión del nervio en el cuello por un manguito traqueal o un tubo endotraqueal grande, en el tórax por cambios en el tamaño de los grandes vasos; estiramiento del nervio a causa de la tracción sobre órganos distantes; efectos tóxicos de sustancias presentes en los tubos endotraqueales o formadas durante la esterilización; y la presencia de una infección concomitante del tracto respiratorio superior.

FIRST INTERNATIONAL SYMPOSIUM ON INTRACRANIAL PRESSURE
to be held in HANNOVER, West Germany, on JULY 27, 28 and 29, 1972.

The meeting will cover three topics:
(1) Methodology of intracranial pressure measurements.
(2) Physiological and pathophysiological aspects of intracranial pressure.
(3) Clinical and therapeutic aspects of intracranial pressure.

Papers submitted will be selected so as to give absolute priority to new data. This will ensure a fruitful meeting. It is intended to leave time for discussions and informal contacts. A scientific exhibit is also being planned on the topic of Methodology. Those interested in it should contact us with details. The modern Hannover Medical School provides excellent conditions for a meeting of this kind.

Correspondence should be addressed to:
M. BROCK, M.D., Neurochirurgische Klinik, Medizinische Hochschule Hannover, 3 HANNOVER-KLEEFELD, Roderbruchstrasse 101, WEST GERMANY.